Posttraumatic Stress Disorder and Negative Symptoms of Schizophrenia

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Posttraumatic stress disorder (PTSD) is highly comorbid with schizophrenia and may be associated with higher levels or lower levels of negative symptoms. In the current study, we attempted to clarify the relationship between PTSD and negative symptoms by examining the proportion of patients meeting various negative symptom criteria in a sample of patients diagnosed with schizophrenia alone or schizophrenia and comorbid PTSD. Results indicated that the presence of PTSD in schizophrenia was associated with increased secondary negative symptoms, with the deficit syndrome (DS) and primary negative symptoms associated with lower rates of current and lifetime diagnoses of PTSD. Furthermore, the deficit/nondeficit classification provided greater differentiation of PTSD symptoms than did negative symptoms defined more broadly using the Scale for the Assessment of Negative Symptoms or primary vs secondary distinctions. These findings suggest that DS patients are at a uniquely low risk for PTSD.

Key words: schizophrenia/deficit syndrome/negative symptoms/trauma/PTSD

Introduction

Anxiety and trauma have long been thought to play a role in the development and maintenance of psychosis. However, few studies have examined the co-occurrence of anxiety disorders and schizophrenia.1 Of the major anxiety disorders identified in the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV),2 lifetime prevalence rates have been estimated to be at least moderate in individuals diagnosed with schizophrenia: obsessive compulsive disorder (1.9%–35.0%), generalized anxiety disorder (2.5%–16.7%), panic disorder (3.3%–20.0%), social phobia (8.2%–36.3%), specific phobia (2.5%–13.6%), agoraphobia (3.8%–6.7%), and posttraumatic stress disorder (PTSD) (19.0%–66.0%).3–5 Of the anxiety disorders, PTSD has received relatively little attention, despite the presumed role of trauma and psychological stress in predicting onset and relapse in schizophrenia.6 Within the limited literature examining trauma and psychosis, individuals with schizophrenia carrying a comorbid PTSD diagnosis have been found to have more frequent violent thoughts, behaviors, and feelings, heightened paranoia, and greater severity of delusions and hallucinations.7–9 Negative beliefs about the self and others may mediate the relationship between trauma and paranoia, and re-experiencing symptoms may mediate the link between trauma and hallucinations.10 Comorbid PTSD has also been associated with a number of poor outcomes, including lower quality of life,5,11 higher rates of suicidal ideation and behavior,12 and increased utilization of medical services.11 Thus, PTSD and trauma seem to have a clear link with functional disability and positive symptoms of schizophrenia.

It is less clear whether symptoms of PTSD are associated with the negative symptoms of schizophrenia, with some studies reporting an increased incidence of negative symptoms13–15 and others reporting decreased negative symptoms.8,16–18 One explanation for these discrepant findings might be that patient samples differed with regard to the prevalence of patients whose negative symptoms could be considered primary or secondary features of the illness. Primary negative symptoms are those that are idiopathic to the disease process itself, while secondary negative symptoms are those that arise from secondary factors, such as depression, anxiety, and medication effects. Patients displaying primary negative symptoms that are persistent for a period of 12 months or longer are further classified as meeting criteria for “deficit syndrome (DS) schizophrenia.”19

If PTSD is associated with increased severity of negative symptoms, one would expect that these negative symptoms would be due to secondary causes. Although not directly stated, such a possibility has been alluded to in previous studies, where it has been suggested that negative symptoms may sometimes occur as an anxiety reaction in response to a traumatic event.1 For example,
it is possible that flat affect may reflect symptoms of emo-
tional numbing often seen in PTSD or that limited social
interaction and poor eye-contact may be indicative of
purposeful avoidance that occurs as part of a PTSD re-
action, rather than symptoms of emotional withdrawal.
In such cases, negative symptoms would not be consid-
ered primary manifestations of the disease process of
schizophrenia itself but rather secondary to symptoms
associated with anxiety and trauma.

It is also equally plausible to expect that patients with
PTSD would be highly unlikely to display negative symp-
toms that are primary in nature. Consistent with this
notion is evidence showing that patients with primary
and enduring negative symptoms, such as those with
the DS, 19 are at reduced risk for experiencing a number
of negative emotions (as measured by clinical ratings)
that are commonly associated with PTSD (eg, depres-
sion, guilt, hostility, and anxiety). 20–24 For DS patients
and for those with primary negative symptoms, it is pos-
sible that the diminished frequency and intensity of
negative emotional experience buffer against the develop-
ment of PTSD when traumatic events are encountered.
Similarly, increased secondary negative symptoms would
be expected when a diagnosis of PTSD is present in
schizophrenia.

At first glance, it would seem that reduced prevalence
of PTSD in patients with primary negative symptoms and
those with the DS would be tautological; however, this is
not the case for several reasons. First, there is mixed
evidence with regard to whether DS patients report expe-
riencing reduced negative affect, with several studies
using clinical rating scales indicating reduction in various
negative emotions 25,26 while other studies using ques-
tionnaires and laboratory-based measures have found
contradictory results indicating that deficit patients actu-
ally have elevated levels of negative emotional experi-
ence. 27–30 Given these mixed results, and the lack of
focused investigations examining primary and secondary
negative symptoms when PTSD is present in schizophre-
nia, it is not currently possible to draw definitive conclu-
sions regarding whether DS patients do in fact have
reduced levels of negative emotion or rather have ele-
vated levels of negative affect and similarly whether their
experience of negative emotions would buffer them from
developing PTSD or put them at higher risk for it. Sec-
ond, it is unclear whether the diminished emotional range
seen in DS patients reflects a reduced capacity to experi-
ence emotion (ie, reduced intensity) or rather a reduced
frequency of emotional experiences in their daily lives.
It may be that when confronted with an emotional stimulus
that carries enough salience, such as a traumatic event,
that event is capable of generating a viable emotional ex-
perience that would not occur during the normal course
of the patient’s life. It is possible that clinician-based
interviews reflect this decreased frequency of emotional
experience, despite the fact that these patients are capable
of experiencing negative emotion when confronted with
salient environmental stimuli. Third, even if patients
with primary negative symptoms experience less nega-
tive emotion at the time of interview/testing (which is
unclear and potentially confounded by methodological
influences), this does not in fact preclude them from
having a lifetime diagnosis of PTSD. It is possible
that DS patients could have developed PTSD before de-
veloping psychosis or before developing negative symp-
toms. It is unknown whether the presumed inability to
experience emotion that is seen in the DS exists prior to
the onset of psychosis or whether such impairments de-
velop later in the course of the illness. As such, it is un-
clear whether DS patients would in fact be at reduced
risk for developing PTSD throughout the lifetime.

Fourth, not all DS patients have diminished emotional
experience. Rather, some have prominent symptoms of
avolition 31 with normal emotional experience when
interviewed using clinical measures such as the Schedule
for the Deficit Syndrome (SDS). Thus, one cannot defini-
tively say that any observed reductions in the rate of
individuals with schizophrenia and comorbid PTSD
who exhibit primary negative symptoms or meet criteria
for the DS is tautological.

Greater knowledge of the characteristics placing indi-
viduals with schizophrenia at risk for PTSD would signif-
icantly advance schizophrenia research and treatment,
allowing for more efficient allocation of intervention
resources and clarification of the effects of trauma on
the course of illness. In the current study, we selected
samples of schizophrenia patients with and without
current/lifetime diagnoses of PTSD and examined the
prevalence of patients within the PTSD groups who
met criteria for various negative symptom classifications.
Individuals with schizophrenia with and without PTSD
were divided into groups based on the severity and cause
of negative symptoms using a number of criteria, includ-
ing (1) high and low negative symptoms defined using
the Scale for the Assessment of Negative Symptoms
(SANS), (2) presence of primary vs secondary negative
symptoms, and (3) presence or absence of DS schizophre-
nia. Comparisons were conducted to determine the prev-
ance of individuals with and without diagnoses of
PTSD who fell into these various negative symptom clas-
sifications. Based upon previous research, it was hypoth-
esized that secondary negative symptoms would be
associated with higher rates of PTSD, while primary
negative symptoms would be associated with decreased
prevalence of current and lifetime PTSD. It was further
hypothesized that the deficit-nondeficit classification
would predict PTSD diagnosis better than negative
symptom classifications made based upon high-low SANS
and primary/secondary classifications alone, reflecting the

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importance of the persistence factor delineated in the DS diagnosis.

Methods

Participants

Participants in the current study included 70 chronic outpatients diagnosed with Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR), schizophrenia.2 Patients were on average 42.4 years old and had 11.9 years of education. The sample was approximately 57.1% male and 45.7% Caucasian. Mean age of onset was 20.4 years. Mean Brief Psychiatric Rating Scale (BPRS)32 total scores were 42.14, indicating that patients were experiencing a moderate level of symptoms at the time of evaluation. Approximately 8.6% of patients were prescribed conventional antipsychotics and 91.4% were prescribed atypical antipsychotics.

Patients were divided into schizophrenia with comorbid PTSD (SZ + PTSD) and schizophrenia without comorbid PTSD (SZ – PTSD) for both lifetime and current diagnoses. For current PTSD diagnoses, SZ + PTSD and SZ – PTSD did not significantly differ on age, education, race, or chlorpromazine equivalent dosage.33 There was a significantly greater proportion of females than males in the SZ + PTSD group. Analysis of the lifetime SZ + PTSD and SZ – PTSD groups also indicated that groups did not differ on relevant demographics, except for a greater proportion of females in the SZ + PTSD group.

Patients were recruited from a local community outpatient mental health center in Nevada and were identified by their treating psychiatrist for inclusion in the study if they carried a primary clinical diagnosis of DSM-IV-TR schizophrenia. All patients were recruited from the community and received monetary compensation for their participation. They were told that they were volunteering to participate in a study examining symptoms, cognition, and emotional functioning. The participation rate of patients referred by treating physicians was moderately high (~70%).

Diagnoses of schizophrenia and PTSD were further confirmed using the Structured Clinical Interview for DSM-IV (SCID).34 Additionally, treating psychiatrists and other program staff were consulted, and medical records were thoroughly reviewed to inform Axis I diagnoses. PTSD diagnoses were supplemented by information gathered from the Life Events Checklist from the Clinician Administered PTSD Scale (CAPS). Exclusionary criteria included (1) English as secondary language, (2) history of traumatic brain injury or condition that would affect central nervous system function, (3) diagnosis of mental retardation, (4) use of medications that could produce significant cognitive effects (other than those prescribed to treat schizophrenia), and (5) corrected vision worse than 20/50.

Measures

Measures included in the current study were designed to evaluate clinical diagnosis, traumatic experience, and schizophrenia symptomology.

Clinical Diagnosis and Symptom Assessment. The SCID34 was administered to determine Axis I psychiatric diagnosis and identify clinical symptoms. All SCID modules were administered; however, only data from the Psychotic and Anxiety disorder modules were examined in the current study. Interrater reliability of the SCID has been shown to be excellent (kappa = .85, range = .71–.97), and diagnostic accuracy, as compared with consensus diagnosis, has been found to be very accurate (82%).35 A study conducted by Fennig et al36 suggests that the SCID-I is a valid instrument for the diagnosis of schizophrenia, as SCID schizophrenia diagnosis displayed good sensitivity (.89), specificity (.96), and agreement (.86) when compared with best-estimate diagnosis made by psychiatrists on first-admission psychotic patients.

The Life Events Checklist from the CAPS37 was administered to all participants. The CAPS is a structured clinical interview measuring major PTSD categories and has been found to have excellent psychometric properties and diagnostic utility.38 The life events checklist assesses whether participants have experienced, witnessed, or learned about 17 common traumatic life events (eg, natural disaster, fire or explosion, serious injury, or harm). Scores are used to estimate the number of traumatic exposures an individual has experienced in a lifetime. In the current study, events endorsed by participants were followed up by questioning to determine whether events were experienced as traumatic and whether events resulted in a traumatic response.

Clinical rating scales completed to assess symptoms of schizophrenia included the SDS,39 SANS,40 Scale for the Assessment of Positive Symptoms (SAPS),41 BPRS,32 and Abnormal Involuntary Movement Scale.42

Procedure

The aforementioned clinical measures were administered as part of a larger battery of symptom, affective neuroscience, and neuropsychological tests. Demographic, diagnostic, and symptom ratings were obtained prior to administration of the neurocognitive tests for all patients. Evaluations typically lasted from 3 to 4 h, and breaks were afforded as needed to diminish fatigue and maintain effort. Evaluations were conducted by experienced doctoral students who were extensively trained to complete procedures in a reliable and valid manner. Evaluations occurred in a quiet and private
setting, and procedures were approved by the University Institutional Review Board.

Classification of Negative Symptom Subgroups

Individuals with schizophrenia were divided into various negative symptom subgroups to determine the association between negative symptoms and PTSD. Three classifications were used: (1) high SANS total vs low SANS total, (2) primary vs secondary, and (3) deficit vs nondeficit. High and low negative symptom classification was determined using a median split on the SANS total score. The high negative symptom group had a mean SANS total score of 22.9 (SD = 10.8) (F = 111.09, P < .001). Primary vs secondary negative symptom distinctions were made using the SDS. Patients were considered to meet criteria for the secondary negative symptom group if they received a score of 2 or greater (moderate severity) on at least 1 of the 6 SDS subscales, and if the symptoms did not meet SDS criteria for being considered primary. Patients who received a score of 2 or greater, as well as a judgment that the symptoms were considered primary on any of the 6 SDS subscales, were designated as falling into the primary negative symptom group. In the event that patients received both primary and secondary ratings for SDS subscales meeting severity criteria, these patients were considered to display secondary negative symptoms. This procedure was implemented to obtain a “purer” group of primary negative symptom patients that would be closer in presentation to DS patients, which would allow for a comparison of deficit vs primary symptoms and, thus, directly assess the effects of the stability criteria of the DS on PTSD comorbidity. This classification resulted in 21 primary and 15 secondary negative symptom patients.

The SDS was used to separate schizophrenia patients into DS and nondeficit syndrome (ND) subgroups. This procedure resulted in the identification of 15 DS and 55 ND schizophrenia patients. DS and ND groups were compared on relevant demographic and clinical features to ensure the validity of the DS classification. Analysis of variance (ANOVA) and chi-square analyses examining variables relevant to DS status suggested that DS and ND patients displayed clinical and demographic features consistent with the DS literature. Specifically, DS and ND patients did not significantly differ on education or age, and both groups had a higher percentage of males than females. There was a trend toward significant differences in ethnicity, where African Americans made up a somewhat greater proportion of the DS than ND sample. DS and ND groups did not differ on age of onset, extrapyramidal symptom severity as measured by the Abnormal Involuntary Movement Scale total score (DS: mean [M] = 7.5, SD = 6.5; ND: M = 5.1, SD = 5.8; F = 1.9, P = .17), or daily antipsychotic medication dosage. DS and ND patients were also prescribed a similar regimen of antipsychotic medication. More severe total SANS negative symptoms and less severe SAPS total positive symptoms were found in the DS group, and no differences were found for BPRS disorganized symptoms (DS: M = 7.3, SD = 2.5; ND: M = 7.4, SD = 3.1; F = 0.01, P = .96). Additionally, DS patients received significantly lower scores on BPRS items assessing dysphoria: Anxiety (DS: M = 1.73, SD = 1.03; ND: M = 3.22, SD = 1.61; F = 11.45, P < .001), Guilt (DS: M = 1.13, SD = 0.52; ND: M = 2.89, SD = 1.76), Hostility (DS: M = 1.27, SD = 0.59; ND: M = 2.30, SD = 1.54), and Depression (DS: M = 1.07, SD = 0.26; ND: M = 1.98, SD = 1.56).

The various negative symptom patient classifications did not differ on age, education, percent of males, ethnicity, chlorpromazine equivalent dosage, or SAPS total scores. Groups were also prescribed a similar regimen of antipsychotic medications at the time of testing. Demographic and clinical characteristics are presented in table 1.

Results

Differences in Negative Symptom Severity Between Patients With and Without PTSD

As a first level of analysis, severity of negative symptoms was compared between schizophrenia patients with and without current and lifetime diagnoses of PTSD. Individual one-way ANOVAs comparing patients with current PTSD (SZ + PTSD) vs patients without current PTSD (SZ – PTSD) on SDS, SANS, and BPRS negative symptom items indicated a significant difference only for the SANS global blunted affect rating, such that SZ – PTSD had a greater severity of blunted affect.

Comparisons of SZ + PTSD lifetime and SZ – PTSD lifetime indicated significant differences for SDS blunted affect, SDS diminished emotional range, SDS diminished sense of purpose, SANS global affective flattening, and BPRS blunted affect. For each of these negative symptom items, SZ – PTSD was found to have a significantly greater severity of negative symptoms (see table 2).

High vs Low SANS Total Negative Symptoms

Current and lifetime PTSD diagnoses were compared among high and low negative symptom groups. Mean percent of high and low negative symptom patients meeting DSM-IV diagnostic criteria for current and lifetime PTSD are presented in table 2. Results of a chi-square analysis for current PTSD diagnosis was nonsignificant, $\chi^2 = 0.76$, $P = .28$; however, the analysis of lifetime PTSD diagnosis approached significance, $\chi^2 = 2.84$, $P < .07$. Additionally, high and low SANS patients reported a similar number of total lifetime traumatic experiences on the Life Events Checklist, $F = 0.20$, $P = .66$ (see table 3).
Table 1. Demographic and Clinical Characteristics of Patient Groups

<table>
<thead>
<tr>
<th></th>
<th>Deficit (n = 15)—Mean (SD)</th>
<th>Nondeficit (n = 55)—Mean (SD)</th>
<th>Test Statistic</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>45.8 (12.5)</td>
<td>41.5 (10.7)</td>
<td>( F = 0.18 )</td>
<td>.19</td>
</tr>
<tr>
<td>Education (years)</td>
<td>12.4 (01.7)</td>
<td>11.8 (01.9)</td>
<td>( \chi^2 = 0.06 )</td>
<td>.52</td>
</tr>
<tr>
<td>Male</td>
<td>60.0%</td>
<td>56.4%</td>
<td>( \chi^2 = 0.10 )</td>
<td>.11</td>
</tr>
<tr>
<td>Ethnicity</td>
<td>26.7%</td>
<td>50.9%</td>
<td>( \chi^2 = 0.67 )</td>
<td>.42</td>
</tr>
<tr>
<td>CPZ dosage</td>
<td>861 (572)</td>
<td>712 (590)</td>
<td>( \chi^2 = 0.64 )</td>
<td>.43</td>
</tr>
<tr>
<td>SANS total</td>
<td>76.3 (23.3)</td>
<td>36.6 (22.8)</td>
<td>( F = 35.1 )</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>SAPS total</td>
<td>21.9 (11.3)</td>
<td>33.6 (19.0)</td>
<td>( F = 5.15 )</td>
<td>.03</td>
</tr>
<tr>
<td>Prescribed conventional</td>
<td>13.3%</td>
<td>09.1%</td>
<td>( F = 0.24 )</td>
<td>.47</td>
</tr>
<tr>
<td>Prescribed atypical</td>
<td>93.3%</td>
<td>90.9%</td>
<td>( F = 0.09 )</td>
<td>.62</td>
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</tbody>
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Primary (n = 21)—Mean (SD)  | Secondary (n = 15)—Mean (SD)  | Test Statistic | P Value |
<table>
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<tbody>
<tr>
<td>Age</td>
<td>46.6 (11.1)</td>
<td>40.5 (7.6)</td>
<td>( F = 3.40 )</td>
</tr>
<tr>
<td>Education</td>
<td>12.1 (01.7)</td>
<td>12.2 (01.8)</td>
<td>( F = 0.01 )</td>
</tr>
<tr>
<td>Male</td>
<td>66.7%</td>
<td>53.3%</td>
<td>( \chi^2 = 0.66 )</td>
</tr>
<tr>
<td>Ethnicity</td>
<td>42.9%</td>
<td>33.3%</td>
<td>( \chi^2 = 0.02 )</td>
</tr>
<tr>
<td>CPZ dosage</td>
<td>811.7 (498)</td>
<td>654 (678)</td>
<td>( F = 0.64 )</td>
</tr>
<tr>
<td>SANS total</td>
<td>53.7 (24.6)</td>
<td>50.1 (25.8)</td>
<td>( F = 0.18 )</td>
</tr>
<tr>
<td>SAPS total</td>
<td>32.5 (19.1)</td>
<td>35.9 (19.2)</td>
<td>( F = 0.28 )</td>
</tr>
<tr>
<td>Prescribed conventional</td>
<td>14.0%</td>
<td>13.0%</td>
<td>( F = 0.60 )</td>
</tr>
<tr>
<td>Prescribed atypical</td>
<td>95.0%</td>
<td>93.0%</td>
<td>( F = 0.06 )</td>
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High SANS (n = 35)—Mean (SD)  | Low SANS (n = 35)—Mean (SD)  | Test Statistic | P Value |
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<tbody>
<tr>
<td>Age</td>
<td>42.8 (12.1)</td>
<td>41.9 (10.3)</td>
<td>( F = 0.10 )</td>
</tr>
<tr>
<td>Education</td>
<td>11.9 (01.7)</td>
<td>11.9 (02.1)</td>
<td>( F = 0.02 )</td>
</tr>
<tr>
<td>Male</td>
<td>62.9%</td>
<td>51.4%</td>
<td>( \chi^2 = 0.93 )</td>
</tr>
<tr>
<td>Caucasian</td>
<td>37.1%</td>
<td>54.3%</td>
<td>( \chi^2 = 4.07 )</td>
</tr>
<tr>
<td>CPZ dosage</td>
<td>885.5 (699.8)</td>
<td>600.3 (500.1)</td>
<td>( F = 3.80 )</td>
</tr>
<tr>
<td>SANS total</td>
<td>67.0 (22.0)</td>
<td>22.9 (10.8)</td>
<td>( F = 11.09 )</td>
</tr>
<tr>
<td>SAPS total</td>
<td>30.9 (16.1)</td>
<td>31.4 (20.4)</td>
<td>( F = 0.13 )</td>
</tr>
<tr>
<td>Prescribed conventional</td>
<td>14%</td>
<td>6%</td>
<td>( F = 0.14 )</td>
</tr>
<tr>
<td>Prescribed atypical</td>
<td>97%</td>
<td>86%</td>
<td>( F = 2.96 )</td>
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Primary vs Secondary Negative Symptoms

PTSD diagnosis was also examined in relation to primary and secondary negative symptoms as rated by the SDS. Results of a chi-square analysis approached significance for current, \( \chi^2 = 3.09, P = .08 \), and lifetime diagnoses, \( \chi^2 = 3.3, P = .07 \). The number of self-reported traumatic experiences as measured by the Life Events Checklist did not differ among patient groups, \( F = 1.2, P = .28 \), indicating that trend-level differences are not accounted for by number of prior traumatic experiences. Thus, there is a trend toward \( \text{SZ} + \text{PTSD} \) being more prevalent in patients whose negative symptoms are secondary vs those whose negative symptoms are primary. The mean percentage of primary and secondary negative symptom patients receiving PTSD diagnoses are presented in table 3.

DS vs ND Schizophrenia

Results of a chi-square analysis examining the relationship between current/lifetime PTSD and DS/ND diagnoses are presented in table 3. Results showed that both current \( \text{SZ} + \text{PTSD} \), \( \chi^2 = 5.21, df = 70, P = .029 \), and lifetime \( \text{SZ} + \text{PTSD} \), \( \chi^2 = 10.95, df = 70, P < .001 \), are significantly less likely to occur in DS than ND patients. Importantly, this reduced prevalence occurs despite a similar number of self-reported traumatic events on the Life Events Checklist, \( F = 0.56, df = 69, P = .46 \). Thus, PTSD is significantly more likely to be associated with the ND then DS form of schizophrenia, even when one accounts for previous traumatic exposure.

Discussion

In our sample of schizophrenia patients, current and lifetime PTSD diagnoses were estimated to be at 21.4% and 44.3%, respectively. These findings are consistent with other studies reporting PTSD comorbidity rates in schizophrenia (19%–66%) and provide further evidence that the occurrence of PTSD in schizophrenia is higher than the rate of PTSD in the general population (8%).

The current findings extend previous research in a number of ways. First, with regard to the mixed findings reported in prior studies, with some indicating increased and others indicating decreased negative symptoms in \( \text{SZ} + \text{PTSD} \), the current results provide tentative evidence supporting both these conclusions. Specifically, when \( \text{SZ} + \text{PTSD} \) patients were classified into high and low negative symptom groups based on a median split from the SANS, results indicated a tendency for lifetime symptoms in SZ.
diagnosis of comorbid PTSD to be associated with less severe negative symptoms \((P = .07)\). Only the global blunted affect item from the SANS proved to differentiate those with and without current PTSD diagnoses. These findings do not support previous studies indicating that comorbid PTSD is associated with greater severity of negative symptoms in individuals with schizophrenia.

In contrast, when patients were divided according to the presence of primary vs secondary negative symptoms using the SDS, there was a tendency \((P = .07)\) for patients with \(SZ + PTSD\) to be more likely to have secondary than primary negative symptoms, providing marginal support for our hypothesis that PTSD results in elevated negative symptoms only in patients whose negative symptoms are secondary. Thus, while these results should be considered preliminary in nature, they do suggest that the mixed results reported in prior studies may be largely due to methodological differences in the classification of

<table>
<thead>
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<th>Table 2. Severity of Negative Symptoms in Patients With and Without PTSD</th>
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<tr>
<td>Current PTSD</td>
</tr>
<tr>
<td>SDS blunted affect</td>
</tr>
<tr>
<td>SDS emotional range</td>
</tr>
<tr>
<td>SDS poverty of speech</td>
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<tr>
<td>SDS curbed interests</td>
</tr>
<tr>
<td>SDS sense of purpose</td>
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<tr>
<td>SDS social drive</td>
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<tr>
<td>SANS global affective flattening</td>
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<tr>
<td>SANS global alogia</td>
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<tr>
<td>SANS global avolition</td>
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<tr>
<td>SANS global anhedonia-asociality</td>
</tr>
<tr>
<td>BPRS emotional withdrawal</td>
</tr>
<tr>
<td>BPRS motor retardation</td>
</tr>
<tr>
<td>BPRS blunted affect</td>
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<tr>
<td>Lifetime PTSD</td>
</tr>
<tr>
<td>SDS blunted affect</td>
</tr>
<tr>
<td>SDS emotional range</td>
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<tr>
<td>SDS poverty of speech</td>
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<tr>
<td>SANS global avolition</td>
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<tr>
<td>SANS global anhedonia-asociality</td>
</tr>
<tr>
<td>BPRS emotional withdrawal</td>
</tr>
<tr>
<td>BPRS motor retardation</td>
</tr>
<tr>
<td>BPRS blunted affect</td>
</tr>
</tbody>
</table>

% Current PTSD | % Lifetime PTSD | Total No. Traumatic Experiences—Mean (SD)

| Table 3. Prevalence of Current and Lifetime PTSD Diagnoses and Mean Number of Self-Reported Traumatic Life Experiences |

<table>
<thead>
<tr>
<th></th>
<th>% Current PTSD</th>
<th>% Lifetime PTSD</th>
<th>Total No. Traumatic Experiences—Mean (SD)</th>
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<tbody>
<tr>
<td>Deficit vs nondeficit</td>
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<tr>
<td>Deficit (n = 15)</td>
<td>0.0</td>
<td>6.7</td>
<td>4.1 (1.6)</td>
</tr>
<tr>
<td>Nondeficit (n = 55)</td>
<td>27.3</td>
<td>54.5</td>
<td>4.9 (4.5)</td>
</tr>
<tr>
<td>Primary vs secondary</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Primary (n = 21)</td>
<td>14.3</td>
<td>23.8</td>
<td>4.4 (3.6)</td>
</tr>
<tr>
<td>Secondary (n = 15)</td>
<td>40.0</td>
<td>53.3</td>
<td>6.1 (5.3)</td>
</tr>
<tr>
<td>High SANS vs low SANS</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High (n = 35)</td>
<td>12.1</td>
<td>34.3</td>
<td>4.5 (4.5)</td>
</tr>
<tr>
<td>Low (n = 35)</td>
<td>25.7</td>
<td>54.3</td>
<td>5.0 (3.6)</td>
</tr>
<tr>
<td>Total</td>
<td>21.4</td>
<td>44.3</td>
<td>4.8 (4.0)</td>
</tr>
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</table>
negative symptoms that fail to consider whether negative symptoms are primary or secondary.

Analyses were also conducted to determine whether individuals with SZ + PTSD were more or less likely to be classified as having DS schizophrenia. Results indicated that patients with SZ + PTSD were significantly more likely to be classified ND than DS. These findings could not be attributed to DS patients having significantly fewer lifetime traumatic experiences, nor could they be attributed to group differences in demographics, severity of positive or disorganized symptoms, or length of illness. Furthermore, the deficit/nondeficit distinction provided greater differentiation of PTSD symptoms than did negative symptoms defined more broadly using the SANS or primary vs secondary classification made using the SDS. The fact that greater separation was found using deficit/nondeficit classifications than high/low and primary/secondary distinctions suggests that negative symptom stability and etiological factors unique to the DS may play a key role in buffering DS patients against the development of PTSD following traumatic exposure. Thus, findings suggest that in schizophrenia, a comorbid diagnosis of PTSD is more closely associated with more transient and secondary negative symptoms and that DS patients are uniquely at reduced risk for developing PTSD. This finding poses the interesting question of: What is it that puts DS patients at reduced risk for PTSD?

The mechanism behind lower prevalence of PTSD in patients with primary negative symptoms and the DS is unclear; however, there are a number of plausible explanations. One possibility is that the diminished experience of negative emotion buffers DS patients from developing PTSD after a traumatic experience. In other words, when confronted with an event that involves actual/threatened death or serious injury, deficit patients may fail to respond with highly intense negative emotion and thereby fail to pair negative affect with a traumatic episode. Similarly, it is possible that impairments in automatically detecting emotional information prevent DS patients from having attention drawn toward salient environmental stimuli that trigger the experience or re-experience of trauma. A third possibility is that DS patients, who have significantly reduced social drive, simply engage in fewer social interactions and, therefore, have lower rates of PTSD because they are exposed to fewer traumatic events. In the current study, this possibility was ruled out as the sole cause because DS patients reported a similar number of traumatic life events as ND patients. A fourth possibility is that neurocognitive impairment precludes DS patients from re-experiencing a salient traumatic event. A recent meta-analysis and new data presented by Cohen et al provided evidence that DS patients display generally reduced neuropsychological functioning when major neurocognitive domains were compared. It may be that impairments in basic attention, working memory, learning, retrieval-based memory, and/or executive functioning reduce the likelihood of having recurrent recollections of an experienced traumatic event or reduce the likelihood that internal or external cues that symbolize the traumatic event will trigger a viable traumatic experience.

The current findings have several limitations. First, generalizability is limited by the somewhat small sample of patients (n = 70) and subsequently reduced power. Our findings may also be limited to samples of chronic outpatients, such as those included in the current sample. Associations between PTSD/trauma and negative/DS symptoms may therefore differ in inpatient and first-episode populations. Additionally, while the SCID has established reliability and validity with regard to diagnosing DSM-IV Axis I disorders, other structured interviews such as the CAPS may have produced somewhat different findings with regard to the diagnosis of PTSD. It is also important to note that a causal inference cannot be drawn from the current findings and that a sequence of the developmental trajectory of PTSD cannot be inferred. It will be important for future studies to collect information regarding the sequence of psychopathology development. To summarize, results indicate that: (1) SZ + PTSD is associated with lower levels of primary negative symptoms, (2) SZ + PTSD is somewhat more likely to be associated with secondary then primary negative symptoms, and (3) individuals with SZ + PTSD are at significantly reduced risk for having DS.

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References